

de Schweinitz (G.E.)

A CASE OF NEURO-PARALYTIC KERATITIS
WITH MICROSCOPICAL EXAMINATION
OF THE DISEASED EYE

BY

G. E. DE SCHWEINITZ, M.D.

OPHTHALMIC SURGEON TO THE PHILADELPHIA AND CHILDREN'S HOSPITALS; OPTHALMOLOGIST
TO THE INFIRMARY FOR NERVOUS DISEASES

[Reprinted from the ARCHIVES OF OPHTHALMOLOGY, Vol. xx., No. 1, 1891]





A CASE OF NEURO-PARALYTIC KERATITIS, WITH MICROSCOPICAL EXAMINATION OF THE DISEASED EYE.

By G. E. De SCHWEINITZ, M.D.,

OPHTHALMIC SURGEON TO THE PHILADELPHIA AND CHILDREN'S HOSPITALS; OPTHALMOLOGIST
TO THE INFIRMARY FOR NERVOUS DISEASES.

SINCE the days when Herbert Mayo showed that section of the trigeminus within the cranium produced insensibility of the eye, and Magendie, in 1824, demonstrated that division of this nerve in rabbits resulted in anæsthesia of the globe, with inflammation and sloughing of the cornea, physiologists, ophthalmologists, and neurologists have undertaken a large amount of experimental research in the endeavor to definitely prove the cause of the corneal lesion. Even at the present day experimenters and clinicians are not in accord, and an examination of the statements in regard to the etiology of neuro-paralytic keratitis found in the standard text-books leaves the reader in doubt whether to accept that theory which ascribes the disease to a trophic change, or that which attributes it to the lessened power of resistance which the cornea, in its insensitive condition, presents to external injuries, or to believe, with Gowers ("Diseases of the Brain," p. 90), that "this neuro-paralytic ophthalmia, as it has been termed, probably depends on the irritation of the nerve by the lesion rather than on the anæsthesia, or on the mere loss of nerve influence."

The case which I am about to detail presents certain interesting features, both in regard to the probable situation of the lesion and the results of the microscopic examination.

William S., a German, aged fifty-eight, was free from disease until the year 1863 (perhaps 1866), when he was attacked, according to his own statement, with a bubo of the right side. He denied having had a chancre, but shortly after the appearance of the bubo his hair fell out. He continued to be in good health until three years later, when he contracted rheumatism, and at the same time iritis, which was double. These conditions were cured, and we have no further record of the disease until March, 1884, when he suffered from left hemiplegia, the arm and leg being involved in the paralysis, with a cure in twelve weeks. In 1885 he had a second apoplectiform seizure, unaccompanied by palsy, and followed by a rapid restoration to health. In January, 1888, he had left facial palsy, at which time the eye was said to have been inflamed, but there is no definite record as to the character of the inflammation, which evidently was not that which subsequently destroyed his sight. In midsummer of the same year he left the hospital without any evident inflammation of either eye, according to the statement of the trained nurses in attendance.

In January of the next year I made the following note :
Right eye—Round pupil ; light reflex preserved ; large, oval, gray optic disc, with dish-like, shallow excavation ; arteries as compared with veins smaller than normal ; spots of pigment on the anterior capsule of the lens indicating the presence of a former iritis. *Left eye*—Pupil nearly occluded by the former iritis, and preventing any view of the fundus ; no evidence of disease of the cornea.

About this time various examinations made by Drs. J. Hendrie Lloyd and Charles K. Mills developed the following facts in regard to this patient's general condition : The left side of the forehead was absolutely smooth, the left angle of the mouth drawn upward, probably due to a spastic condition. There was twitching of the eyelids and muscles of the left side of the face, inability to protrude the tongue, whistle, or dilate the left nostril. The left platysma was weakened, and the masseter and temporal muscles atrophied. The lower jaw could not be protruded to perform grinding movements, sensation was practically lost over the left side of the face, and degeneration reaction present in the muscles supplied by the seventh and fifth nerves. The left arm was paralyzed, with marked atrophy in the extensor and supinator muscles, together with advanced degeneration-reaction and wrist-drop upon this side. The muscles of the left thigh and leg were impaired in power, the extensors and abductors having

suffered more than their antagonists, but no atrophy was present, and there was ready response to both faradism and galvanism. Experiments with the tuning-fork seemed to prove the partial deafness which was present to be due to paralysis of the tensor tympani rather than to disease of the auditory nerve.

In March the type of keratitis which caused the destruction of his eye began to develop, and speedily the following conditions obtained, in spite of all treatment: Coarse episcleral injection, perforation of the lower half of the cornea with hernia of the iris, and a filling of the remains of the anterior chamber with blood, and absolute anæsthesia of the cornea and conjunctiva. Lessened intraocular tension.

To sum up, it will be seen that this patient suffered from an atrophic paralysis with degeneration in the left forearm; a paralysis not complete and not atrophic in the entire left leg; paralysis of the facial nerve and of the motor branches of the fifth nerve, involving some of the sensory fibres, producing anæsthesia of the left side of the face and of the left eyeball.

Dr. Mills, after an exhaustive study of this case, which has been reported in the *Times and Register* of December 7, 1889, and from which the facts in regard to the examinations other than those of the eye have been quoted, came to the conclusion that this man suffered from a lesion in the anterior horn of the left side, in the seventh cervical segment, and that it reached out to the adjoining segments, and also to the white matter of the crossed pyramidal tracts. The condition of the facial muscles was probably explainable by the presence of a lesion of the same character as that in the cord, destroying the cell groups of both the facial and trigeminal nerves.

The left eye, which caused the man great inconvenience and some pain, and the cornea of which had sloughed, was removed, placed in Mueller's fluid, and submitted to microscopic examination. The following lesions were found:

Below the centre of the cornea there is complete perforation of the cornea, the aperture being occupied by the stump of the prolapsed iris, which is granulating and crowded with numerous inflammatory and large, granular, pigmented cells. This central necrosis, which appears to have begun in the true corneal tissue and spread forward until the slough separated and perforation and prolapse followed, is sharply limited on either side, beyond which the cornea is quite free from inflammatory infiltration. As

the periphery of the cornea is reached we find the epithelial layer and overlying conjunctiva much swollen and separated by collections of deeply stained small cells, which have also invaded the corneal tissue, in places massed in pit-like depressions—evidently the earlier stages of abscesses. These lesions do not penetrate the entire thickness of the cornea, and beyond them, passing inward, comparatively healthy tissue remains.

Immediately in the neighborhood of, and surrounding the entrance to, Schlemm's canal, there is a collection of small round cells, apparently extending directly into the lumen of this sinus.

Following the iris from the position of its prolapse toward the ciliary body, this is seen to be swollen, inflamed, and infiltrated with inflammatory cells, a condition which repeats itself in the ciliary body. Of special interest are the vessels in these structures, particularly in the latter. They present one of the forms of obliterating arteritis. Taking as the type a small ciliary arteriole, it reveals in cross-section the following characteristics: An asymmetrically placed lumen contracted out of proportion to the size of the vessel; unusually prominent endothelial plates pushing their way into the central aperture, bounded externally by badly stained fibrous elements that have practically substituted the middle coat, which can still be seen in the periphery as a narrow circle of poorly developed muscular fibres, beyond which the indefinite tissue of the adventitia is apparent. The condition is analogous to that which has been described under the term *mesarteritis*.

The choroid, retina, and optic nerve are free from disease.

The ciliary nerves, where they pass through the sclerotic coat, were carefully sought out, but do not present, in the carmine sections, any evidence of inflammation or infiltration, and in those stained by Weigert's method any sign of degeneration. Their reaction to these reagents was in all particulars such as is seen in nerves of normal constitution.

The principal appearances as found microscopically are hence: A sharply-defined, central slough of the cornea, separated by nearly normal corneal tissue from a peripherally situated, secondary keratitis, having inflammatory connection with overlying diseased conjunctiva, but bounded below by reasonably healthy cornea; small-celled infiltration surrounding Schlemm's canal; inflamed iris and ciliary

body, through which tissues the arterioles present a form of arteritis of the type known as mesarteritis; normal choroid, retina, and optic nerve; and *unaffected ciliary nerves*.

The keratitis present in the eye under consideration, apart from its etiology, is interesting on account of its apparent mode of development. It bears a strong resemblance to the type described by Senftleben (*Virchow's Archiv*, Bd. 65, p. 69), and which he produced in animals in whom the trigeminus had been divided. Under these circumstances this investigator found that the primary affection of the cornea appeared as a necrosis originated by the repeated traumatisms which the eye encountered owing to its anæsthesia. This circumscribed necrosis of the cornea then acted as a seat of inflammatory irritation, and brought into existence a secondary keratitis proceeding from the periphery of the membrane.

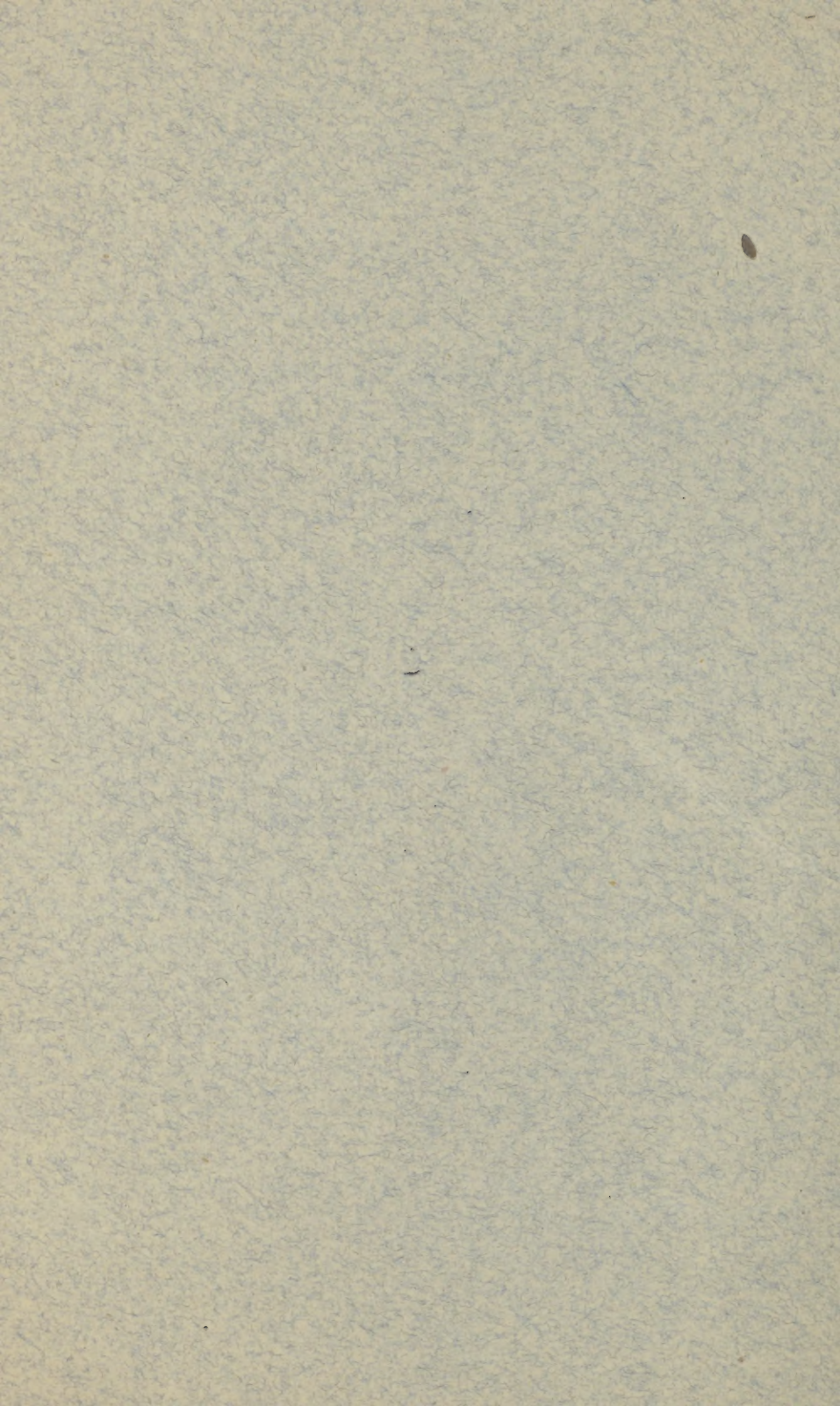
The obliterating arteritis evident in the vessels of the ciliary body, in the study of which I have had the advantage of the assistance of Dr. Meigs, presents an inviting opportunity to construct a theory in regard to the etiology of neuro-paralytic keratitis. As, however, was originally pointed out by C. Friedländer (*Centralblatt f. die med. Wissenschaft.*, 1876, p. 69) arteritis obliterans is found in specimens from clinical and experimental phthisis, in tumors, and in chronic inflammations, especially if accompanied by tuberculosis, an observation which Dr. Meigs has confirmed in his studies of the vessels in various diseased tissues. The existence, then, of this lesion in the present instance is to be explained as the result of a chronic inflammation, and in no sense as a cause of its existence.

The entire absence of disease of the ciliary nerves is a noteworthy fact, and demonstrates that although the nuclei of the fifth pair were affected, there was no degeneration or inflammation in the course of the peripheral distribution of this nerve.

It may be interesting briefly to refer to the various theories which, from time to time, have been advanced to explain the existence of neuro-paralytic keratitis. The earlier experimenters, Magendie, Claude Bernard, and von Graefe,

maintained that the destructive changes in the eye were largely due to the section of a trophic nerve. Snellen concluded that the disease was nothing more than a traumatic inflammation provoked by the presence of undetected injuries and foreign bodies. Schiff, noticing that section was followed by paralysis of the nerves of the vessels, together with widening of their calibre and later inflammation, relegated the cause to the nerve itself through a vaso-motor influence, but afterward modified this opinion by stating that a neuro-paralytic hyperæmia was conditioned by the development of an inflammation stirred up by outside influences. The experiments of Samuel, Buettner, Meissner, Eckhard and others gave rise to the tropho-traumatic theory, Meissner especially holding that the inner fibres of the nerve are more important in the trophic influence than any others. According to Buettner and Meissner the division of these fibres deprived the eye of its capacity to resist external influences. Among other theories is that one elaborated by Eberth, in which a mycotic influence was invoked to explain the disease. The pure traumatic theory, developed originally by Snellen, has found many advocates, notably Senftleben and v. Gudden.

The most recent elaborate work upon this subject is by E. v. Hippel (*Archiv f. Ophthalmologie*, Bd. xxxv., 2, p. 217), from whose paper I have abstracted the facts just quoted. This author cannot accept the presence of medially placed trophic fibres in the trigeminus, believes the pure traumatic theory to be untenable, denies the existence of an impaired power of resisting traumatisms on the part of the affected eye, which, however, is more exposed on account of the anæsthesia to desiccation than is the case with a normal one, rejects micro-organisms as etiological factors, and holds that the theory of increased evaporation is sufficient to explain the development of the inflammation, showing experimentally how a moist atmosphere can prevent the disease.



G. P. PUTNAM'S SONS, PRINTERS
NEW YORK